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Masterclass

Rotator cuff related shoulder pain: Assessment, management and uncertainties

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ABSTRACT

Introduction: Rotator cuff related shoulder pain (RCRSP) is an over-arching term that encompasses a spectrum of shoulder conditions including; subacromial pain (impingement) syndrome, rotator cuff tendinopathy, and symptomatic partial and full thickness rotator cuff tears. For those diagnosed with RCRSP one aim of treatment is to achieve symptom free shoulder movement and function. Findings from published high quality research investigations suggest that a graduated and well-constructed exercise approach confers at least equivalent benefit as that derived from surgery for; subacromial pain (impingement) syndrome, rotator cuff tendinopathy, partial thickness rotator cuff (RC) tears and atraumatic full thickness rotator cuff tears. However considerable deficits in our understanding of RCRSP persist. These include; (i) cause and source of symptoms, (ii) establishing a definitive diagnosis, (iii) establishing the epidemiology of symptomatic RCRSP, (iv) knowing which tissues or systems to target intervention, and (v) which interventions are most effective.
Purpose: The aim of this masterclass is to address a number of these areas of uncertainty and it will focus on; (i) RC function, (ii) symptoms, (iii) aetiology, (iv) assessment and management, (v) imaging, and (vi) uncertainties associated with surgery.
Implications: Although people experiencing RCRSP should derive considerable confidence that exercise therapy is associated with successful outcomes that are comparable to surgery, outcomes may be incomplete and associated with persisting and recurring symptoms. This underpins the need for ongoing research to; better understand the aetiology, improve methods of assessment and management, and eventually prevent these conditions.

1. Introduction

In most cases assessing an individual presenting with a musculoskeletal shoulder problem involves making clinical decisions in highly ambiguous situations (Lewis et al., in press). As part of the assessment process, clinicians need to determine if the symptoms are:

(i) Referred or related to another cause (e.g. from the cervical, thoracic, abdominal regions, neural and vascular tissues)?
(ii) Primarily related to a stiff shoulder (e.g. frozen shoulder, osteoarthritis, locked dislocation, neoplasm-such as osteosarcoma)?
(iii) Due to shoulder instability?
(iv) Related to the soft tissues (e.g. rotator cuff, bursa)?
(v) Due to combinations of the above?

In addition to this, clinicians need to; exclude serious pathology, consider pain mechanisms, determine the relationship and influence of other co-morbidities, and discern the contribution from often profound, obscured and interwoven psychosocial factors. The process is complicated and becomes more so with the emergence of new research information from a multitude of specialities, which is frequently incomplete, and often contradictory.

The purpose of this Masterclass is to focus on one musculoskeletal shoulder problem, rotator cuff related shoulder pain (RCRSP), and discuss; function, pain, aetiology, imaging, surgery, assessment and management.

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2. Rotator cuff function

The rotator cuff (RC) muscles and tendons are commonly considered to be recruited synchronously and equally to dynamically stabilise the humeral head onto the glenoid fossa during shoulder movement. This precept has been challenged and laboratory data suggest that the supra- and infraspinatus are recruited preferentially during shoulder flexion and subscapularis is recruited at higher levels during extension. Higher supra- and infraspinatus activation during shoulder flexion may contribute to reducing anterior glide of the humeral head during activities involving shoulder flexion, and subscapularis activity to reducing posterior glide during extension (Wattanaprakornkul et al., 2011). This asynchronous activity may potentially be used to inform exercise prescription. In addition, in higher ranges of shoulder elevation, such as unsupported abduction, the stabilising function of the cuff may be replaced by the deltoid which may be better oriented to stabilise the humeral head onto the glenoid fossa while allowing the RC to externally and internally rotate the glenohumeral joint (Boettcher et al., 2010). In addition, the belief that supraspinatus initiates abduction appears to be incorrect (Reed et al., 2013).

Clinically, rotator cuff tests have been developed to assess each of the individual tendons by placing the shoulder into defined positions and applying a force to the arm requiring the muscle tissue to contract (Magee, 2014). However the design of the RC does not permit the assessment of an individual musculotendinous unit. The tendons of the RC fuse into one structure, with the supraspinatus and infraspinatus fusing inseparably near their insertion. The muscular portion of teres minor and infraspinatus also fuse inseparably just proximal to the musculotendinous junction. The subscapularis and supraspinatus tendons fuse to form a sheath that surrounds the biceps tendon, and the RC tendons are tightly adherent to the glenohumeral joint capsule (Clark et al., 1990; Clark and Harryman, 1992). Although the interwoven nature of RC, capsule and ligament tissue would improve resistance to failure under load, it negates the possibility of testing individual structural units. The inability to test the RC musculotendinous units in isolation is further evidenced in electromyography, which has demonstrated that during the 'full' and 'empty' can tests designed to implicate supraspinatus pathology, eight to nine other muscles are equally active (Boettcher et al., 2009).

3. Rotator cuff related shoulder pain

RCRSP refers to the clinical presentation of pain and impairment of shoulder movement and function usually experienced during shoulder elevation and external rotation. Although numerous factors including; genetics (Harvie et al., 2004), hormonal influences (Magnusson et al., 2007), lifestyle factors such as smoking (Baumgarten et al., 2010) alcohol consumption (Passaretti et al., 2015), comorbidities and level of education (Dunn et al., 2014), biochemical, patho-anatomical, peripheral and central sensitisation, sensory-motor cortex changes (Lewis et al., 2015) and a raft of psychosocial factors (Dean and Söderlund, 2015) have the potential to contribute to RCRSP, excessive and mal-adaptive load imposed on the tissues appears to be a major influence (McCreesh and Lewis, 2013; Cook et al., 2015).

On-going debate persists pertaining to; (i) the cause of RCRSP, (ii) the mechanism(s) responsible for the sensation of pain, (iii) the relationship between symptoms and structural failure observed within the RC tendons, and (iv) the role and extent of inflammation (Lewis, 2009a, 2011, 2015; Seitz et al., 2011; Lewis and Ginn, 2015; Scott et al., 2015). These symptoms are commonly termed RC tendinopathy which implicates the tendon as the source of symptoms. Although this may be the case, there is no definitive way of incriminating the tendons as the painful structures and the possibility exists that the symptoms are derived from the tendons and their related tissues. As such, and in keeping with other musculoskeletal conditions where a definitive structural diagnosis is often elusive, such as in low back pain (Waddell, 2004), deriving a definitive structural pathognomonic label maybe unachievable, and terms such as; subacromial or rotator cuff pain syndrome or rotator cuff related shoulder pain (RCRSP) may be more appropriate.

4. Aetiology

With respect to mechanism, Neer (1972, 1983) argued that 95% of all RC pathology occurred as a result of irritation onto the subacromial bursa and rotator cuff tendons from the under-surface of the over-lying anterior aspect of the acromion calling the condition; subacromial impingement syndrome. This hypothesis has also been embraced by physiotherapists (Grimsby and Gray, 1997). Once diagnosed, Neer (1983) recommended 12 months of non-surgical treatment for those aged over 40 years with persistent and disabling symptoms, and advocated that if non-surgical treatment was unsuccessful, a subacromial decompression (SAD), also known as an acromioplasty, should be considered. This operation has become one of the most commonly performed musculoskeletal surgical procedures, with a reported 746% increase in the number of SADs in England from 2001 to 2010 (Judge et al., 2014). In England and the USA there has been a concurrent increase in RC repairs. In the USA there was a 141% increase in RC repairs between 1996 and 2006 and embedded within this figure is a 600% increase in repairs performed arthroscopically (Colvin et al., 2012). The reasons for this are uncertain as arthroscopic repairs have been associated with; longer operating theatre times, a higher re-tear rate (46.4%) than open repairs (38.6%) and clinically with no significant difference in outcome (Carr et al., 2015).

The relevance of the acromion to the development of symptoms and RC tears also remains uncertain. The argument that acromial irritation leads to RC pathology is not supported by observational studies. Payne et al. (1997) reported 91% (39/43) of RC tears occurred on the inferior (articular or joint) side of the tendon with only 9% (n = 4) occurring on the bursal side (i.e. the side under the acromion). Fukuda et al. (1987) reported that 82% (n = 27) of tears were joint side or intratendinous and 28% (n = 6) were located on the bursal side. Ellman (1990) reported that from a total of 126 intraoperative investigations of the rotator cuff, 76% (n = 96) were found to have articular side tears, 14% (n = 17) bursal side, and 10% (n = 13) had both. This repeated and consistent finding that tears are predominantly located within the tendon or on the articular side does not support the acromial impingement model. Of relevance, already in 1934, Codman identified articular side tears calling them 'rim rents', stating; '…I am confident that these rim rents account for the majority of sore shoulders. It is my unproved opinion that many of these lesions never heal, although the symptoms caused by them usually disappear within a few months'. In reality, whether these tears cause pain or not, has never been established.

Observed variations in RC morphology may explain some of these findings. Hashimoto et al. (2003) reported greater fibre degeneration and disorientation in the middle and deeper fibres of the RC and argued that degeneration was the primary cause of RC tears. Nakajima et al. (1994) reported that the deeper fibres of the RC had a relatively smaller cross-sectional area than the articular side fibres and the lower fibres failed at approximately half the tensile load of those located superiorly. Bey et al. (2002) reported strain within the supraspinatus tendon that increased with increasing joint elevation between 15° and 60° abduction with no significant difference in strain between the upper and lower fibres.